Clinical Commentary

Equine otitis media-interna

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In the report by Rand et al. (2012), otitis media-interna resulting in meningitis was reported to be uncommon in the horse. Diseases affecting the ear are described according to their anatomical location. Otitis externa involves the external auditory meatus. Equine otitis externa may be bacterial, fungal, neoplastic or parasitic in origin. In practice, otic examinations of horses are rarely done due to the perceived difficulty in visualising the equine external ear canal and tympanic membrane, as well as the need for chemical restraint. Visualisation of the proximal ear canal of the sedated horse can be done with a handheld otoscope but more sedation or general anaesthesia is required to adequately visualise the tympanic membrane in the live horse (Sargent et al. 2006). Otitis media refers to inflammation of the middle ear (also called the tympanic cavity). The middle ear contains 3 tiny bones known as the ossicles: malleus, incus and stapes. These ossicles directly couple sound energy from the ear drum to the oval window of the cochlea. The middle ear is a cavity in the temporal bone from which inflammatory processes may secondarily extend into the inner ear (otitis interna). Otitis interna results in inflammation of the inner ear and bony labyrinth. The inner ear consists of a bony labyrinth where the cochlea is located. The cochlea converts sound pressure impulses from the outer/middle ear into nerve impulses that are transmitted to the brain. In the internal auditory canal, the motor root of the facial nerve (CN VII) and the parasympathetic fibres to the lacrimal glands merge and enter the internal acoustic meatus, accompanied by the cochlear and vestibular divisions of the vestibulocochlear nerve (CN VIII). Lesions in the area of the petrous temporal bone, tympanic bulla and hyoid apparatus can account for clinical signs of otitis media-interna in horses in which the 2 nerves course together closely (Bentz et al. 1997).

Clinical signs of otitis media-interna may include head tilt, nystagmus, falling, circling, ataxia (worsened with blindfolding), muscle deviation, decreased lacrimation, ear paresis, corneal ulceration, inability to blink and depression. Head tilt towards the side of the lesion is common with peripheral vestibulopathy because of the loss of ipsilateral inhibition of muscle tone responsible for head position. Animals also tend to circle toward the side of the lesion. Exaggerated extensor tone may be present in the forelimb contralateral to a lesion that causes vestibulopathy. Relative weakness and resultant ataxia are present ipsilateral to the lesion. These deficits are produced by loss of vestibulospinal tract function. The result is removal of facilitation of the ipsilateral extensor musculature and removal of inhibition of the contralateral extensor musculature (Watrous 1987). Clinically, this can be appreciated with clinical signs of the animal leaning against the wall and falling towards the side of the lesion (Bentz et al. 1997). Recumbent animals tend to lie on the side of the lesion. Pathological nystagmus is involuntary, rhythmic oscillations of the eyes occurring while the head is in a stationary position. Nystagmus with peripheral vestibular disease will be horizontal compared to rotary or vertical nystagmus noted with intracranial involvement. Nystagmus is defined by its fast phase, therefore nystagmus will occur away from the lesion. Ataxia is often present with vestibular dysfunction. The difficulty in distinguishing between proprioceptive deficits and peripheral neuropathy can be overcome with the use of a blindfold. Animals with vestibular disease tend to worsen with blindfolding because of loss of visual compensation (Blythe et al. 1984). If vestibular signs are associated with depression (change in mental state), weakness, seizures and pyrexia then a central vestibular lesion should be suspected.

Facial nerve paralysis (CN VII) is frequently associated with otitis media-interna because of its close proximity to the vestibular nerve. The 2 nerves pass together into the internal acoustic meatus of the petrosal bone. Any disease process of the middle and inner ear could lead to dysfunction of these nerves and thus result in the concurrent paralysis of facial musculature and disturbances of the vestibular system. Paresis or paralysis of the facial nerve results in muzzle deviation away from the lesion, ipsilateral absent menace and palpebral response, ipsilateral ptosis, ipsilateral ear droop and ipsilateral decreased nostril flare. On occasion, decreased lacrimation may be noted because of the disruption of the parasympathetic fibres to the lacrimal gland which travel in close proximity to the facial nerve to the eye (Spurlock et al. 1989).

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The exact pathogenesis of otitis media-interna is unknown but it is currently believed that haematogenous spread of bacteria, ascending infection from the respiratory tract, extension of otitis externa or extension of guttural pouch infection may be responsible for this disorder. Rupture of the tympanic membrane is rarely reported in horses because it is believed that the inflammatory process might extend ventrally to involve the bones of the tympanic bulla and temporohyoid joint. This inflammatory process may result in the osseous proliferation along these bones resulting in the fusion of the temporohyoid joint (Walker et al. 2002). This medical condition is commonly referred to as temporohyoid osteoarthropathy. Progression of the osseous proliferation may lead to impingement and dysfunction of the facial and vestibulocochlear nerves. Alternatively, the stylohyoid or petrous temporal bone may fracture with normal tongue movements resulting in damage of brain tissue or the facial and vestibulocochlear nerves in the acoustic meatus (Hassel et al. 1995). However the theory of pathogenesis of temporohyoid osteoarthropathy remains speculative (Walker et al. 2002). It is possible that cases of temporohyoid osteoarthropathy may be a primary degenerative joint disease rather than an extension of an inner ear infection (Blythe 1997).

The diagnosis of otitis media-interna can be established in horses on the basis of clinical signs, radiographic findings, endoscopic examination of the auditory tube diverticula, computed tomography, results of tympanocentesis and CSF analysis. Radiographic findings may consist of enlargement of the proximal portion of the stylohyoid bone and sclerosis of the petrous temporal bone (Fig 1). Endoscopic examination of the auditory tube diverticula may be a more sensitive procedure than (Hassel et al. 1995) skull radiography for the detection of early osseous changes associated with chronic otitis media/interna (Fig 2). During endoscopic examination osseous proliferation of the temporohyoid joint and enlargement of the proximal stylohyoid bone may be observed. Tympanocentesis is technically a difficult procedure in horses because they have a long external ear canal. This test requires general anaesthesia and thus may make it difficult to justify in terms of risks recovering an ataxic horse. Most clinicians do not perform trans tympanocentesis. However, in the current report by Rand et al. (2012), the horse was showing evidence of central nervous disease that required further diagnostics (CT and CSF collection) that warranted general anaesthesia. It was, therefore, an ideal time to perform the trans tympanocentesis. Trans tympanocentesis is usually done as an adjunctive diagnostic test following radiographic or endoscopic examination. Cleaning the external meatus should be done before general anaesthesia. With the animal tranquillised, the external auditory meatus should be flushed 3–4 times with a dilute solution of iodine and water and the outer ear manually massaged. Multiple rinses with water may be needed to clear the external ear of the iodine with a final rinse of alcohol to help with the drying process. This flushing should be done several hours before anaesthesia. Using an otoscope with a presterilised ear piece the tympanic membrane is visualised. A 6 inch (15.2 cm) spinal needle without a stylet is preloaded with a 3 ml syringe containing 0.75 ml sterile water. Holding the otoscope and external ear in one hand, the operator passes the needle through the otoscope and through the tympanic membrane. Great care must be taken to advance the needle slowly with the directing hand stabilised on something solid. Because there is resistance in passing through the eardrum followed by no or little resistance on entering the air-filled or fluid-filled middle ear, it is easy to injure the mucosa of the middle ear resulting in haemorrhage. Once the needle is through the tympanic membrane, it is advised that another person inject sterile water, then 10–15 s later aspirate back the fluid for culture and fluid analysis (Blythe 1997).

Cerebrospinal fluid evaluation can be performed to determine whether CNS disease is present. On rare occasions, fractures of the temporal bone will result in secondary infection or direct extension of infection may induce sepsis or inflammation of the central nervous system (Blythe et al. 1990).

Treatment of otitis media-interna in horses involves lipophilic antimicrobial therapy for at least 30 days.
that can penetrate the central nervous system. Chloramphenicol and trimethoprim-sulphadiazine are examples of lipophilic antimicrobials that can cross the blood brain barrier. Steroidal and nonsteroidal anti-inflammatory medications are often used to reduce CNS oedema and inflammation. Other medications used for CNS injury in animals may include the use of 10,000 iu a-tocopherol (Vitamin E) per os s.i.d. for 30 days (for 500 kg horse) as an anti-oxidant (Kane 2010). Poor lacrimation and inability to blink may cause exposure keratopathy. The use of eye lubricants or topical antimicrobial treatments (when a corneal ulcer is present) along with partial tarsorrhaphy will be required. Pilocarpine (0.20% concentration, Pilocarpine Hydrochloride ophthalmic solution1), a parasympathomimetic drug that stimulates the lacrimal glands has also been used to aid in increasing tear production (Spurlock et al. 1989). In certain cases (especially relapses) with temporohyoid osteoarthropathy a ceratohyoidectomy may be indicated to help prevent fractures of the petrous temporal bone. However, it is currently impossible to conclude from available data whether or not surgery is appropriate for the prevention of fractures and permanent nerve injury. This author has only used surgical intervention for cases that appeared to have relapsed with regards to vestibulocochlear and/or facial nerve dysfunction.

In a retrospective study involving 33 horses with temporohyoid osteoarthropathy, horses had a fair prognosis (70%) for return to some sort of athletic function. However, the majority of horses would be expected to have some sort of residual cranial nerve dysfunction and it could take a year or longer for maximal improvement to occur (Walker et al. 2002).

**Author’s declaration of interests**

No conflicts of interest have been declared.

**Manufacturer’s address**

1Bausch & Lomb Inc., Tampa, Florida, USA.

**References**


